Summary: Restricted water diffusion has been used to distinguish pyogenic abscess from other rim-enhancing brain masses; however diffusion-weighted imaging of cerebral infection before capsule formation has rarely been described. We report a case of fungal cerebritis in which water diffusion was more restricted than that of normal contralateral brain and the measured diffusion coefficient was in the range of that reported for pyogenic brain abscess. In the proper clinical setting, cerebritis should be considered in the differential diagnosis of an ill-defined focal brain mass associated with markedly restricted water diffusion.

Diffusion-weighted (DW) imaging has been used to diagnose cerebral ischemia in its earliest stages. Restricted diffusion, however, is not specific for acute brain ischemia and has been reported in cases of herpes encephalitis, cortical spreading depression, hyperacute hemorrhage, traumatic axonal injury, Creutzfeld-Jakob syndrome, and, in rare cases, acute multiple sclerosis (1). Abnormal DW imaging has also been reported in cases of pyogenic brain abscess and is attributed to restricted water diffusion in purulent fluid (2). However, MR imaging in earlier stages of brain abscess, before capsule formation, has not been widely reported.

We report a case of cerebritis secondary to frontoethmoidal fungal sinusitis in which restricted water diffusion is demonstrated by use of DW imaging.

Case Report

A 44-year-old woman was found unresponsive and apneic, with twitching of her facial muscles, tongue, and lower extremities. Her family reported that the patient had headache and a supple neck. Blood tests showed an emergency room, initial physical examination revealed a blood pressure of 223/104 and a supple neck. Blood tests showed a diagnosis of insulin-dependent diabetes mellitus and hypertension. In the increasing lethargy of 6-day duration. She had a medical history of suppurative fluid. Ebisu et al (2) were the first to report a brain abscess with marked hyperintensity on DW images; the ADC of in vivo purulent fluid was 0.31 (10⁻³ mm²/s) and of aspirated pus was 0.43 (10⁻³ mm²/s). Subsequent investigators have confirmed

Discussion

Although most commonly used to investigate acute brain ischemia, DW imaging has also helped characterize several focal mass lesions (1). In particular, pyogenic brain abscess may present as a hyperintense mass on DW images because of prolonged T2 relaxation time and because of markedly restricted water diffusion of suppurrative fluid. Ebisu et al (2) were the first to report a brain abscess with marked hyperintensity on DW images; the ADC of in vivo purulent fluid was 0.31 (10⁻³ mm²/s) and of aspirated pus was 0.43 (10⁻³ mm²/s). Subsequent investigators have confirmed
restricted diffusion in suppurative fluid and report ADC values between 0.28 and 0.7 (10^{-3}mm^2/s) (3–5).

Cerebritis is the earliest manifestation of a cerebral infection that may progress to the formation of a brain abscess and occurs 2–3 days following pathogen inoculation in the rat model of cerebral abscess (6). In response to the infecting microbe, an ill-defined area of coagulative necrosis forms with profuse infiltration of the necrotic center by polymorphonuclear leukocytes (6, 7). There is a surrounding area of edematous parenchyma with eosinophilic neurons and blood vessels with a proteinaceous perivascular exudate. Vascular proliferation does not occur until several days later (6). Because it is unusual for patients to present at this stage of cerebral infection, imaging of early cerebritis has not been reported widely. On T1-weighted MR images, an ill-defined area of isointensity or hypointensity and subtle mass effect may be seen, and contrast enhancement is absent or minimal. On FLAIR and T2-weighted images, the infected tissue is hyperintense (8). To our knowledge, only one prior report of diffusion-weighted MR imaging in cerebritis that proceeded to brain abscess formation exists in the literature (9). The case we present is important, because it shows that early cerebritis should be added to the growing list of ill-defined focal lesions that may be associated with restricted water diffusion.

In the absence of purulent fluid, restricted diffusion in early cerebritis might be attributed to hypercellularity, brain ischemia, or cytotoxic edema. Because the translational movement of water occurs primarily in the extracellular compartment, increased cellularity from the abundant infiltration of neutrophils may restrict water diffusion because of the reduced extracellular space, a more complex intracellular environment, or both. For example, hypercellular high-grade primary brain neoplasms typically have a lower diffusion coefficient compared with that of normal brain tissue (10). When a contiguous extracerebral infection such as frontal sinusitis exists, cerebritis may develop from retrograde diploic or emissary thrombophlebitis (7). Although many cases of occlusive venous ischemia are characterized by increased water diffusion from vaso- genic edema, some cases are associated with reduced diffusion (11). Although not observed in our case, mycotic infections may infiltrate along cerebral vessels, causing a necrotizing angiitis and arterial thrombosis (12). In these cases, cytotoxic edema from arterial ischemia may explain restricted water diffusion. Finally, restricted diffusion in cases of herpes and other viral

Fig 1. Images acquired on hospital day 5 during the early stage of left orbitofrontal cerebritis.

A, FLAIR image (8000/105 [TR/TE]; TI, 2500) shows hyperintense subcortical white matter of left frontal lobe (arrow).

B, Postcontrast T1-weighted image (650/17) shows no contrast enhancement around an ill-defined hypointense area (arrow). Note mucosal enhancement in left frontal sinus (white arrow).

C, DW image (b = 1000 s/mm^2) shows hyperintense signal (arrow) in frontal cerebritis.

D, ADC map shows hypointense signal intensity (arrow) indicating restricted water diffusion. Mean ADC is 0.41 ± 0.04 (10^{-3}mm^2/s).
FIG 2. MR images acquired on hospital day 9 during the late stage of cerebritis or early stage of abscess.
A, Contrast-enhanced T1-weighted image (650/17) shows thin, faint peripheral contrast enhancement around a left frontal mass (arrow).
B, DW image (b = 1,000 s/mm$^2$) demonstrates marked hyperintensity with a rim of even higher signal intensity (arrow).
C, Restricted diffusion is indicated by hypointensity (arrow) on this ADC map. Mean ADC is 0.59 ± 0.03 (10$^{-3}$mm$^2$/s).

FIG 3. Images acquired before abscess aspiration on hospital day 15.
A, FLAIR image (9000/105; TI; 2500) shows a poorly defined left frontal mass (arrow).
B and C, Postcontrast axial (B) and coronal (C) T1-weighted images show a thin, well-defined enhancing wall, consistent with cerebral abscess.
D, DW image (b = 1000 s/mm$^2$) shows marked increased signal intensity is present in this mass (arrow).
E, Marked hypointensity (arrow) on ADC map is consistent with restricted diffusion. Mean ADC is 0.56 ± 0.05 (10$^{-3}$mm$^2$/s).
encephalitis has been attributed to direct cytotoxicity that results in neuronal swelling (1).

Conclusion

We report a case of cerebritis secondary to fronto-ethmoidal fungal sinusitis in which water diffusion is more restricted than that of normal contralateral white matter and the measured diffusion coefficient is in the range reported for that of brain abscess. Cerebritis should be considered in the proper clinical setting when an ill-defined focal brain mass is associated with markedly restricted water diffusion.

References


Fig 4. Images acquired after surgical aspiration and antifungal therapy; cerebral abscess has resolved, leaving focal gliosis.

A, FLAIR (7000/105; TI, 2500) shows hyperintense signal in the left frontal lobe (arrow).
B, Postcontrast T1-weighted image (650/17) shows a band of contrast enhancement at the site of treated abscess (arrow).
C, DW image (b = 1000 s/mm²) shows symmetric signal intensities in frontal lobes.
D, ADC map shows minimal hyperintensity (arrow) in left frontal lobe, a finding consistent with gliosis. Mean ADC is $1.87 \pm 0.08 \times 10^{-3} \text{m}^2/\text{s}$. 

A, FLAIR (7000/105; TI, 2500) shows hyperintense signal in the left frontal lobe (arrow).